16.

A CASE OF LESION OF THE OPTIC THALAMUS WITH AUTOPSY

BY

GORDON HOLMES, M.D.

Assistant Physician to the National Hospital, Queen Square, and to the Seamen's Hospital, Greenwich

AND

HENRY HEAD, M.D., F.R.S.

Physician to the London Hospital

[Reprinted from "Brain," 1911, Vol. XXXIV, p. 255]

JOHN BALE, SONS & DANIELSSON, LTD.
OXFORD HOUSE
83-91, GREAT TITCHFIELD STREET, OXFORD STREET, W.

1912

A CASE OF LESION OF THE OPTIC THALAMUS WITH AUTOPSY.

BY GORDON HOLMES, M.D.,

Assistant Physician to the National Hospital, Queen Square, and to the Seamen's Hospital, Greenwich,

AND

HENRY HEAD, M.D., F.R.S.,

Physician to the London Hospital.

Since the publication of Roussy's monograph on the "Syndrome thalamique" in 1907, very few cases with the clinical features and the anatomical lesions which he identified with this syndrome have been recorded. The only ones of which we know are the two further ones described by Roussy himself, two by Long [4], and one by Winkler and van Londen [10]. These, with Roussy's three original observations, and cases recorded by Dide and Durocher [1], Roque, Chalier and Cordier [7], and Paillard and Lelièvre [6], make a total of eleven cases recorded since attention has been directed by Dejerine to this type of disease of the optic thalamus. A search amongst the earlier literature, however, shows that similar cases, in which the site of the lesion was confirmed by autopsy, had been published by Edinger [2], Greiff [3], and others.

All these cases conform, more or less closely, to the clinical description given by Dejerine and Roussy, and justify its recognition as a clinical type, though even Roussy's own later experiences have shown that in some respects the definition he gave it was too rigid. The absence of a severe hemiplegia with contractures, and of an extensor response, were among the characteristic features he originally described, but they were present in one of his later cases. On the other hand, involuntary choreo-athetoid movements were originally regarded as a typical feature, but they were absent in two of Roussy's own cases.

Despite these discrepancies there can be no doubt that these cases represent a definite clinical group, and, as *post-mortem* examination has shown, their more characteristic symptoms depend on destruction of a special and definite region of the optic thalamus, that is, the posterior part of its external nucleus.

On the other hand, it must be recognized that this syndrome, which Dejerine and Roussy have described most fully, is probably associated with lesions of this portion of the thalamus only, and that disease of other parts of this mass of grey matter may produce essentially different clinical phenomena.

The following case, in which we had the opportunities of full clinical and pathological investigations, evidently belongs to the Dejerine-Roussy group.

W. H., aged 49, was admitted to the National Hospital on August 23, 1909, into one of Dr. Ormerod's beds, but owing to Dr. Ormerod's absence he was, whilst in hospital, under the care of one of us (G. H.). He was a blacksmith, and had until recent years been in good health. He denied alcoholic excess and venereal infection.

The history he gave of his illness was confirmed and amplified by his wife. The first evidence of any cerebral disease was a temporary difficulty in speech in February, 1906, from which he recovered completely within three days. He had an attack of "influenza" in the autumn of 1908, but was soon able to resume his work, at which he remained till early in 1909, when he had to cease owing to vertigo and a feeling of general weakness. He had complained since then constantly of these symptoms.

In February, 1909, while talking to his wife, he suddenly fell to the ground and lost the power of movement in his right limbs and the right side of his face, but there was no loss of consciousness. He recovered rapidly and was soon again able to use the right limbs quite well. In June, 1909, he had another similar attack, and a third on July 1. The right limbs were weaker after each of these attacks, but he remained able to move them, though he could not employ his right hand in feeding himself, or in other fine actions. Speech was not noticeably affected and the patient's general mental condition did not alter materially. From the date of the first attack, however, he complained much of headache and vertigo.

On the evening of the day on which he was admitted to hospital he became maniacal and violent and had to be removed to the isolation ward. Next day, however, he was quiet, collected and easy to examine. His mental state improved further and for a few days he remained in a condition fairly suitable for our investigations.

He was a sallow, lean man with marked arcus senilis, rigid arteries and a high pulse tension; his heart was hypertrophied, and his urine was of low specific gravity and contained a small amount of albumen. He appeared much older than his age. His memory was poor, and, as he was inclined to become inattentive after a time, it was necessary to interrupt the examination frequently. There was no trace of aphasia or apraxia.

His ocular movements were normal: the pupils were equal but small, and inactive to light. The movements of the jaw were unaffected, and there was no

weakness or asymmetry of the face in either volitional or emotional movements. His palate and tongue moved straight, and there was no alteration of articulation, phonation, or deglutition. Visual acuity was R. $\frac{6}{24}$, L. $\frac{6}{12}$ but there was no hemianopia. Ophthalmoscopic examination, however, revealed severe disease of the retinal arteries, and occlusion of the upper temporal artery of the left eye. Associated with this vascular disease were œdema, hæmorrhages and extensive degeneration of the retina, sufficient to account for the visual deficiency. Hearing, too, was defective, and according to his wife it had been so since the stroke in February, 1909. He was very deaf in his left ear, but on the right side he could hear the tick of a watch by aerial conduction, but not through the bone.

Motor system.—The left limbs were unaffected.

The muscles of the right arm were equal in size to those of the left, but their tone was slightly increased, though the limb could not be described as rigid. Its various movements were unrestricted in range and there was no tendency to contractures. It was difficult to estimate the actual power of the limb as, owing probably to the sensory loss, he seemed unable to exert a constant effort with it; but there was very little, if any, true weakness as compared with the left arm. All its movements were, however, slow, awkward and ataxic, even when controlled by vision, and extremely erratic when his eyes were closed.

There was also slight increase of tone of the muscles of the right leg, but no tendency to contractures or limitation of movement. The same difficulty was met with as in the arm in estimating the strength of the limb, but there seemed to be little or no weakness. Its movements were very awkward and ataxic. He walked with short, uncertain steps, holding the right leg rigidly extended as though it were spastic.

Reflexes.—The deep reflexes were all brisk but equal on the two sides. The abdominal and cremasteric reflexes were also equal, and the plantar reflexes of both sides were characteristically flexor in type.

Spontaneous sensations.—It was difficult to obtain from the patient a full description of his sensations, but he complained constantly of pains and soreness in his right arm, chiefly in the hand and forearm, and in the right foot. These pains were "as if you were bending up the arm and crushing it," or "as if you were breaking it." He also complained of sharp shooting pains in the right side, which usually started in the leg and shot up this side of the trunk into his head; he would frequently grasp his head between his hands and shriek out with pain. He suffered also with "an aching, crampy feeling" in his right limbs and theeright side of his body.

Tactile sensibility.—He failed to recognize ordinary cotton-wool touches on the right limbs and on the right side of his trunk, though he responded immediately to even the lightest touches on the left side. Occasionally he appreciated contacts "faintly" on the hair-clad parts of the right side of his face, but not on hairless regions, as on the forehead. On the trunk the anæsthesia to cotton-wool extended almost to the middle line, but by using

slighter stimuli, as a von Frey's hair of 23 grm./mm.^{2}, it was found to extend at least 1 cm. beyond it. When a wisp of cotton-wool was rubbed repeatedly and firmly over hair-clad parts of the limbs he could occasionally recognize the stimulus, but vaguely and "faintly." No tactile hairs up to 100 grm./mm., could be appreciated anywhere on the right arm, the right side of his trunk, or on the right leg, but contacts with a stimulus of this strength could be felt on the right foot, where the sensory loss was certainly less profound than on the rest of this side. Similarly, firm touches with the pulp of a finger or with any blunt object could not be recognized on the right side, except on the foot and the lower part of the leg.

Corneal sensibility.—When tested with von Frey's hairs it was found that, while on the left cornea even 12 grm./mm.^{2} evoked a sensation and a considerable reaction, on the right no hair below 21 grm./mm.^{2} produced a sensation, and even this excited only a slight reflex.

Localization of contact could not be tested owing to the gross defect of tactile sensibility, but in testing painful sensibility it seemed that the patient could localize, at least approximately, pricks that evoked pain.

Compasses.—Records could be obtained from the soles only, as this was the only part of the right side where contacts could be appreciated when the points were applied with reasonable pressure. A perfect series of answers was obtained on the left sole with the points 5 cm. apart, but on the right sole he failed constantly to recognize the double nature of the stimulus even when the points were separated to 10 cm.

Vibration.—He could only appreciate vibration on the right limbs and the right side of the trunk when the fork (C. 128) was vibrating strongly, and when it had apparently ceased to vibrate on this side he could feel the vibrations again for a considerable period if the fork was immediately transferred to the corresponding part of the left side. The diminution of sensibility was less on the leg and foot than elsewhere on the affected side. When asked to compare the sensations evoked on the two sides he said the vibrations "felt louder" on the left than on the right side.

Sensibility to pain.—There was almost complete insensibility to pin-pricks on the right arm and on this half of the trunk; it was impossible to evoke pain even by pricking him repeatedly on the one spot with a sharp pin. On the trunk the analgesia extended to within 1 cm. of the middle line, but apparently pricks did not become naturally sharp until the middle line was crossed. The right side of his glans penis was also insensitive to prick.

On the right leg, especially on its distal segments, pricks could evoke pain, but to do so they had to be stronger than was necessary on the opposite side; the threshold was certainly considerably raised. The pain evoked was generally not so severe as that produced by a similar prick on the left foot, but it was accompanied by a greater reaction.

The greater part of the right side of the head was insensitive to prick, but on the cheeks, lips, and on the forehead just above the eye, even gentle pin-pricks evoked pain.

It is noteworthy that a series of pricks on the one spot of the right arm would occasionally start a movement, though the patient showed no discomfort, and as a rule did not intimate, even when questioned, that he was aware of any stimulus.

Pressure-pain.—It required considerably more pressure with the algometer to elicit pain on the right than on the corresponding parts of the left side, but the discomfort that was then produced, and the reaction it caused, were unquestionably greater on the affected side. A striking feature of these observations was that the pain seemed to appear suddenly or explosively and did not develop gradually as it did on the normal side. The following figures give the approximate pressures that evoked pain on the two sides:—

	R.			L.		
Palm	15	12	11	8	8	8
Temporal fossa ..	5½	4	3½	2	2	2
Sole	9	9	10	5	4	4½

Thermal sensibility.—On the right hand and arm there was complete loss of the appreciation of temperature as such, and the patient reacted only to the disagreeable elements of extreme temperatures. Stimuli between 20° C. and 55° C. evoked absolutely no sensation, but when tubes below 20° C. or above 55° C. were applied to the hand or arm they produced a very intense reaction, the patient withdrawing his hand vigorously, screwing up his face as if in extreme pain, and crying out, "Oh, something has caught me," or "Something is forcing its way into me," or "It's got hold of me; it's pinching me."

The loss of thermal sensibility was very similar on the right side of the face, though he seemed to recognize heat as such, when the temperature was relatively high, in the neighbourhoood of the eye. There was also complete loss on the right leg, but on the sole temperatures above 55° C. gave him a sensation of heat; ice, on the contrary, evoked only "a crampy pain," which spread up his leg.

The painful sensations evoked by hot tubes and ice persisted for a considerable time after the stimulus was removed, and according to the patient's description spread widely from the spot stimulated. A tube at 55° C. applied to the sole, for instance, produced a pain that radiated "all over the foot." Equally striking was the fact that the application of high or low temperatures had to be of a considerable duration, occasionally as much as thirty seconds, before the pain developed; then, like the pain produced by pressure, it appeared explosively.

Sense of position of the right limbs was completely abolished; he had no idea how or where they lay when his eyes were closed. This was easily demonstrated by requesting him to place his left (sound) forefinger on his right hand or foot, or to indicate the position of the latter by pointing with the left hand; in seeking his right arm he merely waved the left about until he found his shoulder, and then traced the arm to the hand.

Appreciation of passive movement was also completely lost in the right limbs; he was, in fact, unable to recognize any change of position of these limbs at any of their joints.

Appreciation of weight.—A fairly good threshold could be demonstrated for the left hand, but even the presence of 100 grm. could not be recognized on the right hand, whether it was supported or unsupported.

Other sensory faculties, as the ability to recognize size, shape, and form, could not be tested owing to the serious disturbance of tactile sensibility.

The above notes were completed on September 1. The patient's condition, both mental and physical, then deteriorated so much that no further systematic investigations could be carried out, but we succeeded in confirming many of the facts recorded above.

He died on September 9, and a complete autopsy was performed eleven hours later.

Post-mortem Examination.

The heart was considerably enlarged, owing chiefly to hypertrophy of the left ventricle, but its valves were normal. There was remarkably little disease in the aorta, but many of the smaller vessels were thickened and atheromatous. There was some hypostatic congestion of the bases of the lungs, but they were otherwise normal. The other viscera were free from disease, excepting the kidneys which were small, shrunken and firm; the capsules when stripped off left an extremely granular surface, and on section the cortex appeared very narrow. On microscopical examination the usual features of a small cirrhotic kidney were discovered.

The spinal cord appeared large and well formed. There was no meningitis or other disease visible on the surface, and no degeneration could be detected by the naked eye in its cross-section.

The meninges of the brain were normal except for a slight thickening at the base and on the lips of the Sylvian fissures. The internal carotid and the vertebral arteries were thickened, irregular in lumen and atheromatous, and the larger branches of the circle of Willis, excepting the anterior cerebrals, were similarly diseased. The brain itself was firm, large and well developed. No softening or other disease could be detected on its surface. It was immediately placed uncut into 10 per cent. formalin in normal saline solution for preliminary hardening.

Microscopical Examination.

The spinal cord was examined at various levels by both Marchi's and Weigert's stains, but no systemic degeneration or other disease could be detected in it by either method.

The brain-stem.—When the brain was hardened by formalin the

brain-stem was separated off by a transverse section through the anterior quadrigeminal bodies. One thin slice taken from this region, and another from the middle of the bulb, were treated by Marchi's method; the rest was examined in almost serial sections by Weigert's stain.

In the sections of the mid-brain prepared by Marchi's method there was visible in the middle third of the left pes, in the ordinary position of the pyramidal tracts, a slight amount of degeneration. There was also secondary degeneration of some of the dorsolateral fibres of the left dorsal longitudinal bundle, and a considerable amount of degeneration in the whole cross-section of the mesial fillet. The latter was characterized by a remarkable fineness of the Marchi granules. No degeneration was visible in the other tracts of the mid-brain. In the Marchi sections of the medulla oblongata only a few degenerated fibres were visible in the dorsomedial portion of the left pyramid and some degeneration, represented by very fine granules, in the interolivary layer. On the other hand, no degeneration or disease could be detected in the sections stained by Weigert's method.

There can be no doubt that the slight degeneration of the pyramidal tracts revealed by Marchi's method was due to involvement of certain of its fibres by the lesion that lay on the mesial side of the internal capsule. The degeneration of the mesial fillet contrasted with this by the much finer Marchi granules that represented it, and it is certainly to be regarded as a retrograde or cellulo-petal change, due to the affection of the distal portions of its fibres at or near their termination in the optic thalamus, rather than a true secondary or Wallerian degeneration. This retrograde atrophy or degeneration of the fillet has been repeatedly observed after such lesions. This view is confirmed by the fact that the systematic examination of the rest of the bulb failed to reveal any local lesion that could account for secondary degeneration of the fillet.

The fore-brain.—The fore-brain was cut by sections in the plane of the optic tracts into slices about 1 cm. in thickness. To the naked eye no disease was visible in the right hemisphere, or in the cortex or subcortical white matter of the left, but a focus of softening was at once detected, which appeared in the more posterior sections immediately on the inner side of the external geniculate body and extended from here forwards, through the lateral portion of the optic thalamus, adjacent to the internal capsule.

Those blocks which contained the cerebral end of the mid-brain, the optic thalamus and the corpus striatum, as well as the greater part of

the rest of the left hemisphere, were then mordanted in Müller's fluid, embedded in celloidin, cut in serial sections and stained by Weigert's or by the Weigert-Pal method. The accompanying photographs show the site and extent of the disease that was discovered, and consequently a short description will suffice.

FIG. 1.—Section through the level of the posterior commissure. The softening, as it appeared under the microscope, is outlined by a broken line. Another small focus of softening lies in the dorsal part of the pulvinar.

The caudal extremity of the lesion, which extended just below the section shown in fig. 1, lay immediately mesial to the external geniculate body and to the dorsolateral angle of the pes, but neither of these structures was definitely affected by it. It spreads mesialwards into the inferior portion of the pulvinar, but not further ventralwards than the level of the posterior commissure. A little higher, at the upper

end of the external geniculate body (fig. 1) it lies chiefly in the ventral portion of the pulvinar, but its lateral angle projects into the lamina medullaris externa immediately dorsal to the crus. At this level the mesial fillet, as it bends dorsolateralwards into the thalamus, lies immediately ventral to the lesion, but is not here directly involved by it. It appears, however, poorly stained and atrophied. There is also slight rarefaction in the outer third of the pes, but this is otherwise normal.

Fig. 2.

The lesion occupies the same relative position in the succeeding sections but becomes larger. In a section through the ganglion habenulæ and the corpora mammalaria (figs. 2 and 3), its main portion lies ventral to the anterior end of the pulvinar and extends mesialwards to about the plane of the lamina medullaris interna, and ventralwards into the ventral nucleus. Its lateral portion at first projects almost

horizontally outwards, breaks through the lamina medullaris externa into the reticular zone of the thalamus, and there spreads in the plane of the section along the inner margin of the internal capsule. The latter, however, is not directly invaded by the softening, and no degeneration can be detected in it by Weigert's method.

At this level, and slightly above it, that portion of the ventral nucleus of the thalamus in which the fillet terminates is invaded by

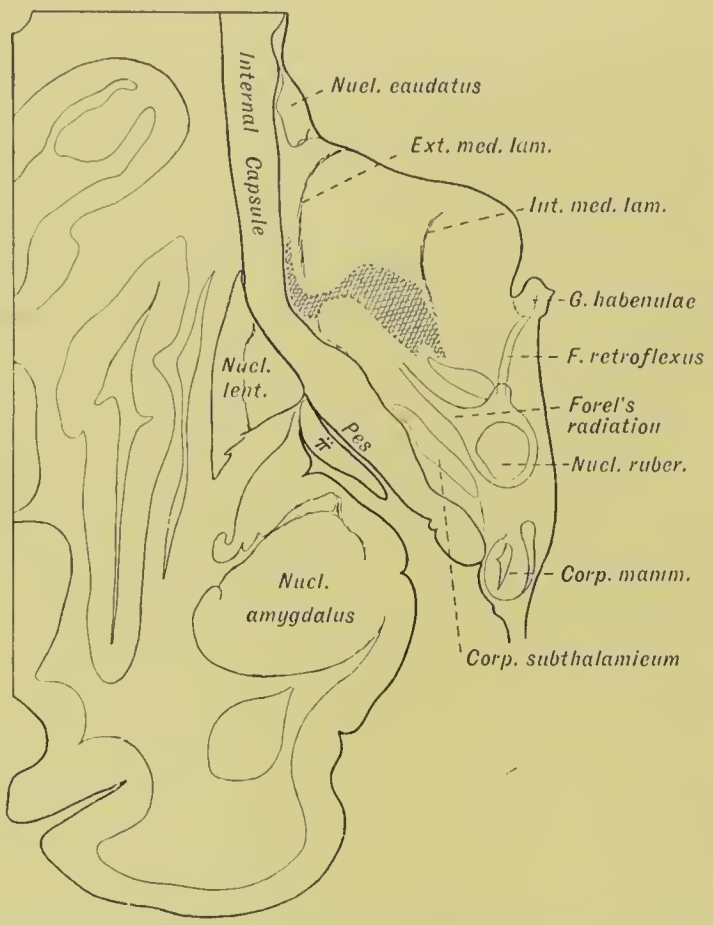

FIG. 3.—A drawing of the section shown in fig. 2, under a lower magnification. The lesion, as seen under the microscope, is cross-hatched.

the lesion. To this, and more especially to involvement of some of its fibres, the retrograde atrophy of the fillet must be attributed.

As the lesion is traced upwards its ventromesial portion becomes smaller, while the lateral part is larger (fig. 4), and extends vertically through the lateral zone of the thalamus. The mesial part extends inwards to the lamina medullaris interna, but leaves the mesial nucleus almost intact; while its lateral portion forms a narrow strip in the reticular zone, extending dorsolateralwards almost to the caudate

nucleus, and ventromesialwards to the lateral angle of the corpus subthalamicum. At this level in fact it runs more or less parallel with the internal capsule, and separates the thalamus from it. It maintains this form and position almost to the dorsal surface of the thalamus, but in the highest sections of this (fig. 5) it is not quite continuous, but is represented by two distinct foci in the reticular zone and the lamina medullaris externa.

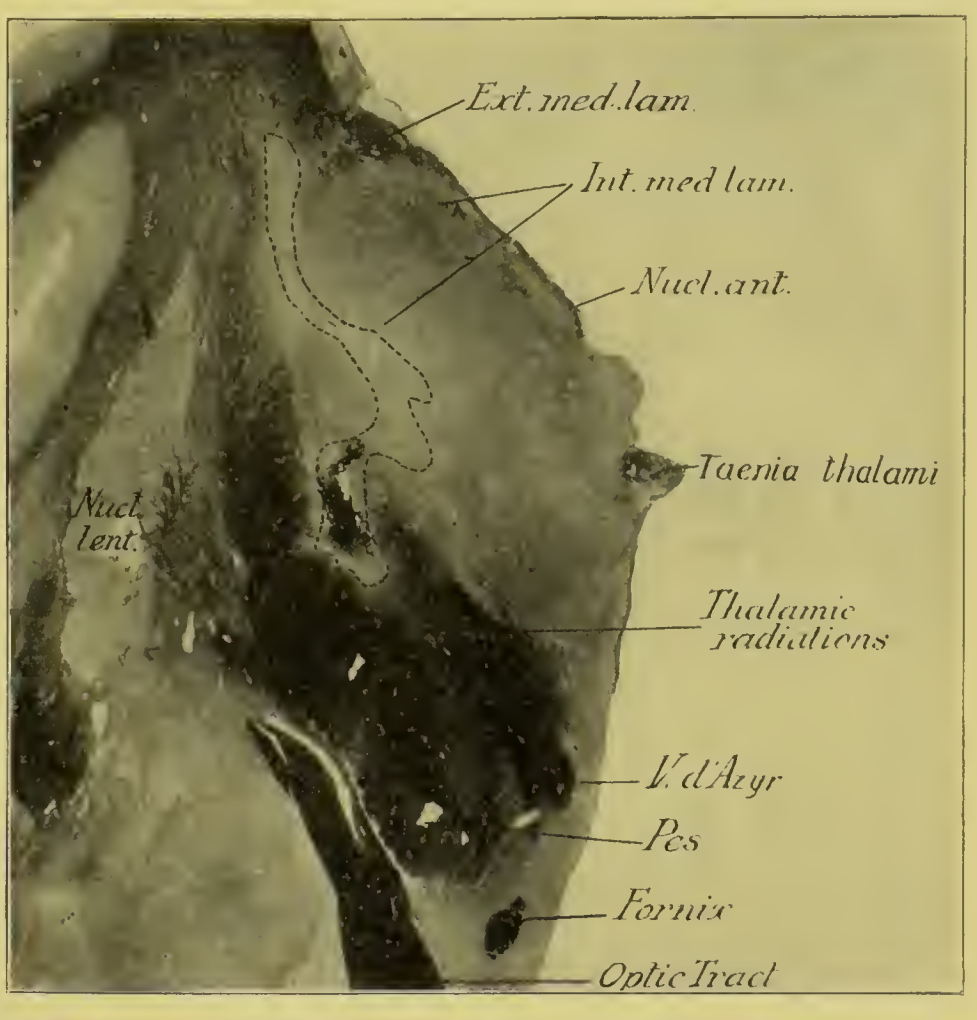

Fig. 4.—The lesion is outlined. It contained at this level in its ventral part a relatively recent hæmorrhage.

The internal capsule was not actually involved by the lesion at any level, though here and there its fibres appeared rarefied where the lesion impinged upon it.

The lesion consisted of a softening with complete destruction of the cells and fibres within it, and their replacement by loosely proliferated neuroglia. Its limits were easily definable, but in places it appeared as

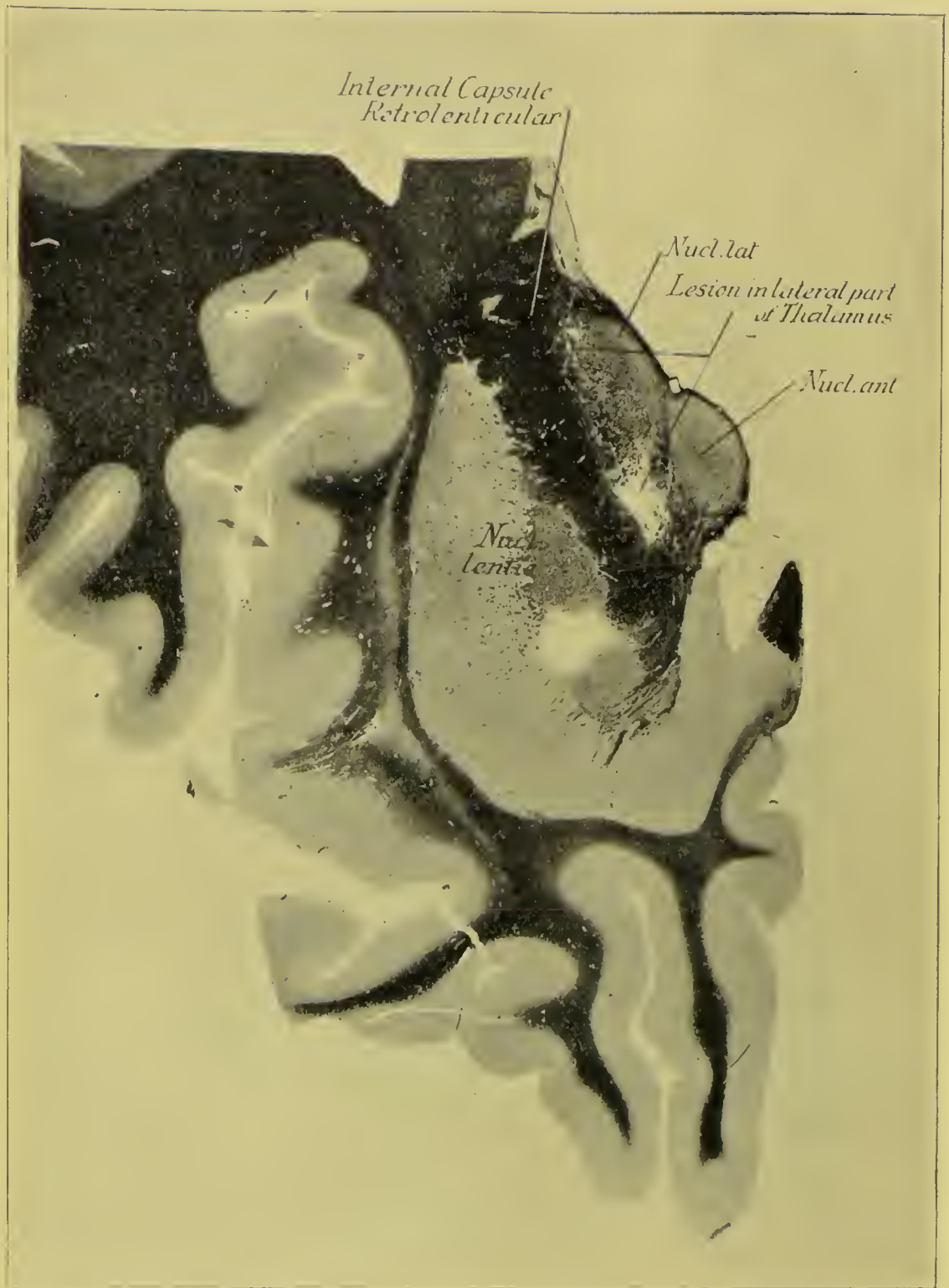

FIG. 5.—The softening in the lateral nucleus is here represented by two foci. A small patch of softening also lies in the nucleus lenticularis.

if the vascular occlusion that produced it had caused a necrosis of tissue more extensive than the actual softening. It was consequently necessary to outline its actual extent in the photographs that serve as illustrations. In one place (fig. 4) the softening contained traces of a relatively recent hæmorrhage.

The only other focus of disease, excepting a minute softening in the dorsal part of the pulvinar (fig. 1), was a small patch of softening, shown in fig. 5, in the lenticular nucleus on the outer side of the genu of the internal capsule.

The cortex of the central convolutions was examined by Nissl's method, but no definite pathological changes were discovered.

We can *summarize* this case as follows: A man, aged 49, with chronic Bright's disease and hypertrophy of the heart, suffered from three strokes at short intervals, without loss of consciousness, the last more than two months before his death. There was little or no paralysis, and the reflexes remained normal, but he suffered with severe pains throughout the whole of his right side, associated with almost complete loss of tactile and postural sensibility on this side. The threshold for painful stimuli was much raised, though certain forms of discomfort, when evoked, were more severe, less bearable, and produced a greater reaction. The appreciation of temperature was abolished, but thermal stimuli below 20° C. and above 55° C. produced more discomfort than on the normal side, and an excessive reaction. There was also loss of the ability to appreciate weight, size, shape and form. He had bilateral deafness, greater on the side opposite to the lesion, as a result of the strokes.

On *post-mortem* examination an extensive softening was found in the ventral and lateral regions of the left optic thalamus, which invaded that portion of the ventral nucleus of the thalamus in which the fillet terminates. The greater portion of the softening, however, formed a narrow cleft in the lateral zone of the thalamus separating it from the internal capsule. The internal nucleus of the thalamus was very little affected.

It is evident that both the clinical symptoms and the anatomical lesion in this case conform closely to those of the type described by Dejerine and Roussy. It will be therefore interesting to collate the symptoms we observed in this patient with those of similar cases, in which the site and extent of the disease was determined by autopsy. For this purpose we may refer particularly to those recorded by Edinger,

Greiff, Roussy (five cases), Long (two cases), and Winkler and van Londen. The cases published by Roque, Chalier and Cordier [7], and by Paillard and Lelièvre [6], ended in death so early after the onset of the lesions, that the effect of shock on their symptomatology cannot be excluded. In that recorded by Dide and Durocher [1] the exact extent of the lesion was not determined by microscopical examination.

Roussy defined the "syndrome thalamique" by the following symptoms and signs: hemianæsthesia, involving deep sensibility more than the superficial forms, with persistent, paroxysmal, spontaneous pains on the affected side; little or no hemiplegia; hemiataxia, and frequently irregular involuntary movements of the athetoid or choreic type, in the affected limbs.

Our case presented all these symptoms except involuntary movements, but on analysing the ten cases referred to we find that this symptom was present in four only of them (Roussy [8], obs. 1, and [9], obs. 1; Edinger and Greiff). Consequently, it cannot be regarded as an essential clinical feature of this group, though it is undoubtedly highly characteristic when it occurs. Hemiataxia, which was pronounced in our case, is apparently a more frequent symptom; it was definitely recorded in seven of the ten cases.

The sensory loss in our case was greater than in most of those we refer to, but in this respect it closely resembles one of Roussy's ([9] obs. 1). The sensory disturbance, however, speaking merely of these ten cases, is very variable in its intensity and, as Roussy has shown, affects chiefly what he calls "deep sensibility," that is, the postural elements. In another of his cases, for example ([8] obs. 3), the appreciation of touch, pain and temperature was unaffected, and the sense of position and the recognition of form alone were lost. In Edinger's case, on the other hand, the sense of position was undisturbed, and there was only an over-reaction to certain stimuli.

Spontaneous pains and paræsthesiæ, which were a prominent feature in our case, are more constant and characteristic, and were, in fact, absent in two only of the ten cases (Roussy [8] obs. 2, and Winkler and van Londen).

But there is another clinical feature of these cases which has not hitherto attracted special attention, though its occurrence has been frequently mentioned in the clinical descriptions; this is a tendency for certain stimuli, generally of the unpleasant order, to produce more pain and discomfort on the affected than on the normal side

of the body, despite the diminution of sensibility. In our own case almost all stimuli that could evoke a sensation had this effect; pricks when they affected consciousness, it is true, produced only a greater reaction, but sensibility to this form of stimulation was very low. With painful pressure, however, the excessive discomfort and reaction were very pronounced; while high and low temperatures, which were recognized only as painful or unpleasant stimuli, had the same effect. Roussy refers to this phenomenon as "dysesthésie." In his first case ([8] obs. 1) prick evoked numbness and disagreeable sensations: there was "hyperæsthesia" to cold, and even the tuning-fork produced a sensation of "intense burning." In a second case ([8] obs. 2) prick produced a painful numbness that was badly localized, and heat above 50° C. evoked pain. In another case ([9] obs. 1), he observed that tickling was extremely unpleasant to the patient, and in his last recorded case ([9] obs. 2) even stroking the skin or hairs was painful, and thermal stimuli evoked tingling. In Edinger's case even touch was painful and cold was unbearable, and in Greiff's hyperæsthesia was observed. Winkler and van Londen found that pinching or squeezing evoked "a much sharper perception of pain" on the affected than on the normal side, and this was associated with "vehement repelling movements." In fact, in two only of these ten cases, in those recorded by Long, was there no mention made of this tendency for affective stimuli to evoke pain and discomfort.

We have discussed elsewhere the significance of this phenomenon, which we regard as the most constant and characteristic feature of lesions of the lateral zone of the optic thalamus, and need not enter on it here again. We may only mention that we discovered it in some form or other in all the twenty-four cases of thalamic lesions that we have studied clinically.

Another symptom in our case, unhappily one that was not carefully investigated, was a partial deafness which, according to the history, supervened on the attacks that produced his other symptoms. It was bilateral, but greater on the side of the lesion. Owing to the incomplete examination of the ears we cannot exclude local or other causes of this deafness, but as it has been observed in other cases it deserves a short reference. In two of his cases ([8] obs. 1, and [9] obs. 2), Roussy merely records slight diminution of hearing on the same side as that affected by the other symptoms, and no further analysis of the nature of the defect was made. In Winkler and van Londen's case almost complete deafness followed on the lesion, but hearing

returned partially; and in Greiff's case hearing was diminished on both sides. We also found defective hearing in several of the clinical cases we have investigated; in some its occurrence was definitely associated with the stroke that produced the other symptoms, and its nature suggested that it was of central origin. The most interesting communication on this point is that published by Merle [5]; for in his case partial bilateral deafness, greater on the side of the sensory symptoms, followed immediately on the stroke, and as in a case that we have observed, was associated with extreme susceptibility to loud sounds and noises, which became actually painful to the patient. This phenomenon, as Merle points out, is analogous to the over-response to certain sensory stimuli that also occurs in these cases.

The affection of hearing has been generally bilateral, though usually greater on the side opposite to the lesion. Its occurrence is easily explicable by the site of the lesion, for when situated in the posterior and lateral part of the optic thalamus it may involve the internal geniculate body itself; or, as in Winkler and van Londen's case, it may interrupt the cortico-petal fibres from the internal geniculate body as they pass through Wernicke's zone.

Finally, we may point out that the disease in our case occupied approximately the same site as the lesions in other cases with similar symptoms. According to Roussy, the characteristic lesion destroys the posterior third of the lateral nucleus, and parts of the internal nucleus, of the centre median and of the pulvinar; and these were the portions of the thalamus that were actually affected in our case. But perhaps its most striking feature was that the internal capsule and the crus escaped almost entirely despite the size of the lesion; in this respect it resembles most closely the cases of Edinger, and of Winkler and van Londen. The thalamic lesion in our case, too, was probably larger and more extensive than in any instance yet recorded, as it reached from the lateral geniculate body to the nucleus anterior thalami; and yet it left the internal division of the thalamus almost entirely intact.

REFERENCES.

[1] Dide et Durocher. "Un Cas de Syndrome thalamique avec Autopsie," *Rev. neurol.*, 1904, t. xii, p. 802.

[2] Edinger, L. "Giebt es central entstehende Schmerzen?" *Deutsche Zeitschr. f. Nervenheilk.*, 1891, Bd. i, S. 262.

[3] Greiff, F. "Zur Localisation der Hemichorea," *Arch. f. Psychiatr.*, 1883, Bd. xiv, S. 598.

[4] Long, E. "Deux Observations anatomo-elinique de Syndrome thalamique," *Rev. neurol.*, 1910, t. xix, p. 197.

[5] Merle, P. "Syndrome thalamique et Troubles auditifs," *Rev. neurol.*, 1909, t. xvii, p. 1528.

[6] Paillard et Lelièvre. "Syndrome thalamique," *Bull. et Mém. de la Soc. anatom. de Paris*, 1910, t. lxxxv, p. 790.

[7] Roque, Chalier et Cordier. "La Syndrome thalamique," *Lyon médicale*, 1908, t. cx, p. 1054.

[8] Roussy, G. "La Couche optique," Paris, 1907.

[9] *Idem.* "Deux nouveaux Cas de Lésion de la Couche optique, suivis d'Autopsie," *Rev. neurol.*, 1909, t. xvii, p. 301.

[10] Winkler and Van Londen. "On the Ventral Group of Nuclei in the Thalamus Opticus of Man," *Proc. Koninklijke Akademie van Wetenschaffen*, Amsterdam, 1908, vol. ix, p. 295.